

# Abdominal Wall Biloma: An Unusual Complication of Laparoscopic Cholecystectomy

Jaco H. Festekjian, MD, S. Ahmad Hassantash, MD, Edward W. Taylor, MD

## INTRODUCTION

Laparoscopic cholecystectomy (LC) was first described in 1988 by Reddick,<sup>1</sup> Dubois,<sup>2</sup> and Perissat.<sup>3</sup> With improvement in laparoscopic instrumentation and increased expertise of surgeons, LC has rapidly become the procedure of choice for the treatment of symptomatic cholelithiasis.<sup>4</sup> Unfortunately, LC is associated with a higher rate of complication than open cholecystectomy. The incidence of extrahepatic biliary tract injury is reported to be 4% in early series and 0.3% in more recent reports.<sup>5</sup> This is still about three times higher than the 0.125% bile duct injury rate for open cholecystectomy (OC).<sup>6,7</sup> Cystic duct leak represents 19% of biliary tract injuries.<sup>8</sup> Diagnosis and evaluation of a leak site may be made by HIDA scan of the biliary tree. Some biliary leaks may be managed expectantly. Drainage of the bile collection followed by close observation often serves as definitive therapy.<sup>9</sup> However, some bile leaks require a more aggressive form of intervention for the eventual resolution. The most common form of intervention is endoscopic retrograde cholangiopancreatography (ERCP) with or without biliary endoprosthesis placement. Some patients will require concomitant sphincterotomy. The second most common form of intervention is reoperation (open or laparoscopic) and re-ligation of the cystic duct stump.<sup>10</sup> This approach is more commonly utilized for early or persistent leaks. Between 1989 and the time of this report, we have performed 1024 LC's. In this report, we describe an unusual post-LC complication which was managed nonoperatively.

## CASE REPORT

A 35 year old obese (250 lbs) female underwent LC for symptomatic cholelithiasis. She had no signs or symptoms to suggest choledocholithiasis. Liver function tests were normal. She did not have a history of any prior abdominal operations. The operative technique utilized is that described by Reddick with minor variations.<sup>11</sup>

CO<sub>2</sub> insufflation of the peritoneal cavity was obtained via a Veress needle successfully placed infraumbilically during

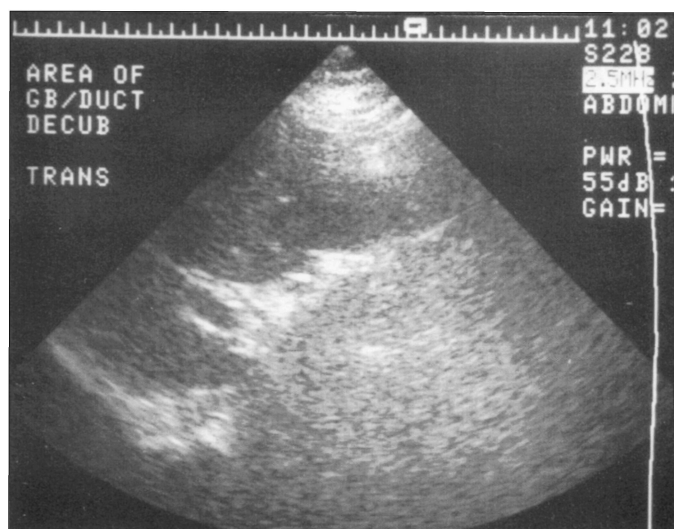
the first attempt. A 12 mm trocar was introduced infraumbilically without complication. Dissection of the cystic duct was conducted very close to the neck of the gallbladder (GB). We were not able to cannulate the cystic duct and elected to proceed with LC without cholangiogram. We routinely attempt intraoperative cholangiogram but in this case the anatomy was clear, the patient had no positive indicators for choledocholithiasis and the cholangiogram catheter would not thread. Before completely dividing the GB from its bed, a last look in the triangle of Calot revealed continued but minimal biliary staining close to the cystic duct stump. No GB injury had occurred during the dissection and the cystic duct clip looked secure. Because the anatomy was quite clear, and our dissection was confined to the GB neck, we did not suspect a common bile duct injury. However, with biliary staining despite complete irrigation, we elected to leave a closed suction drain in the GB fossa. The infraumbilical fascia was closed with absorbable suture and the patient was admitted to the hospital for observation.

During the 24-hour period of in-hospital observation, no biliary drainage was observed. The patient was afebrile, anicteric, and had mild (appropriate) postoperative, incisional tenderness. She tolerated a regular diet, the drain was removed, and she was discharged home. On the third postoperative day, the patient was readmitted to the hospital with the complaint of right upper quadrant pain and biliary drainage from the infraumbilical incision. She did not report fever, chills, nausea, or vomiting. Her vital signs were normal. She was anicteric and had a benign abdominal examination. Her infraumbilical incision was draining bilious fluid. Her white blood count was 14,000 per mm<sup>3</sup> (normal: 4.0-11.0), 80% (normal: 45-70%) of which were segmented neutrophils. Liver function parameters were within normal limits. Suspecting an intra-abdominal bile collection, we obtained an abdominal ultrasound on the day of readmission. This failed to show any intraperitoneal fluid collections (**Figure 1**). The biliary tree was not dilated and common bile duct measured 3.3 mm in diameter. Intravenous antibiotics were started and the patient was observed in the hospital.

During the patient's three day hospital stay, her vital signs remained stable. Her abdominal pain slowly resolved. The biliary drainage from the infraumbilical incision (a total of

Department of Surgery, Kern Medical Center, Bakersfield, California.

Address reprint request to: Edward W. Taylor, MD, FACS, Department of Surgery, Kern Medical Center, 1830 Flower Street, Bakersfield, CA 93305, USA. Telephone: (805) 326-2274, Fax: (805) 326-2282

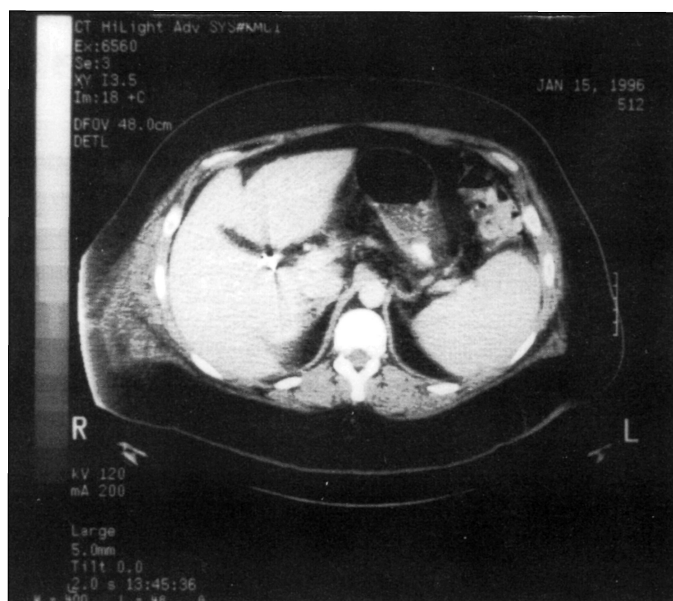


**Figure 1.** Abdominal ultrasound done in 3rd postoperative day illustrating the lack of fluid collection in the Morrison's pouch and normal extrahepatic biliary tract.

250 cc in three days) ceased spontaneously. On the fifth postoperative day, there was a sudden, spontaneous onset of drainage from the right lateral superior 5 mm trocar site (the same site that the drain had exited). The drainage, approximately 200 cc, resolved quickly over several hours. Interestingly, physical examination revealed a well-circumscribed greenish staining of the abdominal wall. This area was nontender, without any fluctuance. A computerized tomographic (CT) scan of the abdomen and pelvis was performed on postoperative day six. This revealed a nondilated biliary tree, and no fluid collection in the peritoneal cavity. However, there was a large fluid collection present in the abdominal wall with the following boundaries: from T11 superiorly to L3 inferiorly, and from the anterior axillary line anteriorly to the posterior axillary line posteriorly (**Figure 2**). This collection measured 7.5 cm by 15 cm with a maximum thickness of 5 cm.

In the hospital, the patient's total and conjugated bilirubin rose to a high of 2.2 and 1.4 mg/dl, respectively (normal: 0.0-1.5 and 0.0-0.4 respectively). Over the next 36 hours, her mild hyperbilirubinemia resolved. Her serum Alkaline Phosphatase, AST, and ALT levels were never elevated.

At the time of discharge from the hospital, she was asymptomatic. No interventional attempt was made to drain the collection. Her vital signs remained within normal limits. The greenish discoloration of her skin persisted until about postoperative day 14. She tolerated a regular diet and had an uneventful recovery. The patient was followed closely as an outpatient for another two months. A follow-up CT scan of the abdomen and pelvis showed a normal liver and biliary tree, no intraperitoneal collection, and resolution of the abdominal wall fluid collection.



**Figure 2.** Abdominal CT scan demonstrating the fluid collection in the abdominal wall, which is located both superficial and deep to the abdominal wall musculature.

## DISCUSSION

Biliary leak is known to be one of the more common complications of LC. The incidence is reported to be about 0.4%.<sup>5</sup> Common causes of bile leak include: cystic duct leak, common bile duct or hepatic duct injury, or a GB bed leak (ducts of Luschka). The first reported biliary leak following LC was presented by Dubois.<sup>2</sup> Among 36 LC patients presented in this series, they report a case in which the patient complained of abdominal pain on the second postoperative day. Ultrasound detected a fluid collection suggestive of a bile leak. At laparotomy there was no leak from either the cystic duct or the common bile duct. The leak was presumed to be from the liver bed and the area was drained. In our patient, we noticed a small amount of bile staining but no extrahepatic biliary tree injury was identified. The clips on the cystic duct seemed to be secure. Although presence of an injury to the extrahepatic biliary ducts or leak from the cystic duct can not be completely ruled out, the most probable source was the GB bed (ducts of Luschka). The low output of bile, (<500 cc in the next postoperative week), and early discontinuance of the leak, both support this theory.

Another interesting aspect of this case is the accumulation of bile in the abdominal wall yet ultrasound and CT scan failed to show a fluid collection in the peritoneal cavity. Whether the drain was functioning in the first 24 hours or not, the leaking bile must have been either collecting in the peritoneal cavity or already started to have access to the

abdominal wall. In the former scenario the fluid must have drained into the abdominal wall within the next two days to account for a lack of peritoneal fluid on ultrasound. In contrast to open cholecystectomy, in which all abdominal wall layers are closed, the defects created by trocars in LC are left open (except the infraumbilical incision). Also, CO<sub>2</sub> insufflated into the peritoneal cavity during LC can escape the peritoneal cavity and accumulate in the abdominal wall thus creating pseudo-pockets thus facilitating fluid collection. From the location of the biloma, we assume that the upper lateral incision was responsible for the entry of bile into the abdominal wall. The proximity of the wound to the biliary ducts and/or the GB bed and the fact that the drain exited this incision, may be contributing factors.

We treated this complication nonoperatively as the patient had a relatively benign clinical presentation. The liver function parameters were within normal limits. Minimal transient increase in bilirubin was probably due to absorption of bile from the patient's abdominal wall collection. Not all patients with bile leaks present with such a benign postoperative course and ERCP or HIDA scan may facilitate prompt diagnosis and guide timely treatment. However, this case demonstrates that in selected circumstances, conservative nonoperative management without the use of invasive diagnostic tests or therapeutic intervention can have a favorable outcome.

## References:

1. Reddick EJ, Olsen DO. Laparoscopic laser cholecystectomy. A comparison with minilap cholecystectomy. *Surg Endosc.* 1989;3:131-133.
2. Dubois F, Icard P, Berthelot G, Levard H. Coelioscopic cholecystectomy: preliminary report of 36 cases. *Ann Surg.* 1990;211:60-62.
3. Perissat J, Collet D, Belliard R. Gallstone-laparoscopic treatment-cholecystectomy, and lithotripsy our own technique. *Surg Endosc.* 1990;4:1-5.
4. Schirmer BD, Edge SB, Dix L, et al. Laparoscopic cholecystectomy: Treatment of choice for symptomatic cholelithiasis. *Ann Surg.* 1991;213:665-676.
5. Ress AM, Sarr MG, Nagorney DM, et al. Spectrum and management of major complications of laparoscopic cholecystectomy. *Am J Surg.* 1993;165:655-662.
6. Henry ML, Carey LC. Complications of laparoscopic cholecystectomy. *Surg Clin North Am.* 1983;63:1191-1204.
7. Strasberg SM, Hertl M, Soper NJ. An analysis of the problem of biliary injury during laparoscopic cholecystectomy. *J Am Coll Surg.* 1995;180:101-125.
8. Woods MS, Traverso LW, Kozarek RA, et al. Characteristics of biliary tract complications during laparoscopic cholecystectomy. *Am J Surg.* 1994;167:27-32.
9. Soper NJ, Flye MW, Brunt LM, et al. Diagnosis and management of biliary complications of laparoscopic cholecystectomy. *Am J Surg.* 1993;165:663-669.
10. Woods MS, Shellito JL, Santoscoy GS, et al. Cystic duct leaks in laparoscopic cholecystectomy. *Am J Surg.* 1994;168:560-565.
11. Reddick EJ, Frasier W. Laparoscopic cholecystectomy: a report on 500 consecutive cases. *Surg Laparosc and Endosc.* 1991;1:2-7.